



Management of Pilonidal Sinus with Ksharsutra Ligation Introduction

Presented by-

Dr Sanjeet Kumar Sahu

Dr. Shubham sharma

(PG Scholar VYDS Ayurvedic Mahavidyalaya Khurja UP)

Guided by-

1. Prof. Dr. Amit Mishra (HOD of Shalya Department, VYDS Ayurvedic Mahavidyalaya

Khujja Bulandshahr UP.

2. Dr. Mandar taru (Professor of Shalya Department., VYDS Ayurvedic Mahavidyalaya Khurja UP.

3. Dr. Tejaswini Jagannath Haryan (Assistant Professor of Shalya Department, VYDS

Ayurvedic Mahavidyalaya khurja , UP

Introduction

Pilonidal disease was originally described by Herbert Mayo in 1833 as a congenital condition with the term 'pilonidal', derived from the Latin 'nest of hairs', being coined by Richard Hodges in 1880 [1]. Diagnosis was through identifying a characteristic epithelial track (the sinus) located in the skin of the natal cleft. During the Second World War the condition was common in jeep drivers, hence the term 'jeep disease' [2,3]. Moreover, a similar condition was identified in the interdigital clefts of clefts of barbers caused by hair entering moist, damaged skin [4]. Current understanding acknowledges pilonidal disease as an acquired chronic infection of the natal cleft skin and subcutaneous tissue that manifests acutely or with intermittent symptoms over several years.

Pilonidal disease has a reported incidence of 26 per 100,000 in the US [5] and 48 per 100,000 in Germany [6]. The condition is more common in Caucasians due to hair characteristics and growth patterns [5,7], typically affecting the teenage to young adult population up to the 3rd decade. The mean age of presentation is 21 years in men and 19 years in women [8]. Furthermore, the prevalence amongst men is two to three times that of women [9,10]. Therefore, pilonidal disease represents a significant disease burden, affecting people in their most productive years with huge socioeconomic implications. In its most severe form, pilonidal disease can be severely debilitating, causing daily discomfort and limiting activity.

A wide range of treatment options have evolved with rates of recurrence and morbidity from traditional surgical approaches unacceptably high. This review evaluates current and future treatment modalities considering the evolving understanding of disease pathophysiology. These highlight the need to critically re-evaluate the surgical treatment of pilonidal disease and embrace newer treatment modalities. Moreover, non-surgical treatment for uncomplicated pilonidal disease is gaining popularity where efficacy needs to be evaluated in light of current evidence

Etiology

Pilonidal disease was thought to be of congenital origin, but increasing evidence indicates an acquired aetiology [11,12]. Firstly, occupation plays a major role with reports of occurrences between the fingers of sheep shearers, dog groomers, and barbers [4]. Further risk factors include, a sedentary lifestyle, positive family history, obesity, hirsute body habitus, local irritation or trauma [5,11]. Secondly, blocked hair follicles can lead to enlargement and rupture of the pilosebaceous glands

with either abscess formation or a chronically discharging sinus [13]. In addition, Bascom postulated pilonidal disease as originating from a stretched midline hair follicle of the epidermal skin layer, analogous to an epidermal inclusion microcyst, thereby advising against resecting deep tissue during surgery [7]. However, Karydakos reported loose hairs, burrowing into otherwise normal tissue, inducing a foreign body reaction leading to secondary pits and cyst formation [14]. The source of the hair can either be the natal cleft itself in hirsute individuals, or hair from the head or back that falls into the natal cleft. The hair follicle becomes distended and obstructed leading to oedema and inflammation. Subsequently, a chronic abscess may develop, with a track draining it known as a sinus [12,15]. Furthermore, epidermal and deep tissue disruption are amplified by changes in the cleft microenvironment including increased moisture, anaerobic environment and bacteria in the natal cleft. Anaerobic bacteria (Bacteroides and Enterococci) predominate in the development of follicular infection and abscess formation and subsequent wound breakdown following surgery [16,17]. However, in 49 postoperative wound complications, aerobic bacteria was isolated in 43% of cases vs 40% anaerobic isolates [17]. Moreover, preoperative antibiotic usage did not show reduction in the wound complication or recurrence rate after 30–42 months followup [17,18]. Therefore, the role of bacteria in initiating, persisting and recurrent pilonidal sinus disease evolves with disease progression and host response.

These factors have implications for both the extent of disease expression and progression. This was incorporated in a mathematic model following review of over 6000 patients [12]. The three primary variables were [1]: loose hair or “invader” (H) applies some [2] force (F), which is influenced by secondary factors such as the depth, narrowness, and friction of the natal cleft to create an insertion process. The third factor of vulnerability, (V), refers to the local tissue susceptibility. In this model, the primary sinuses represent the hair entry sites and secondary sinuses represent the exit points [19]:

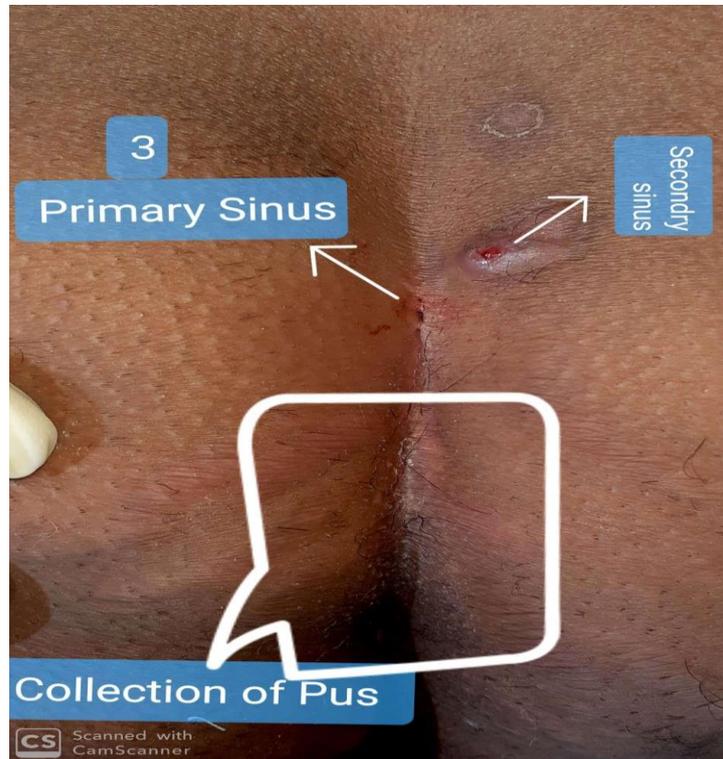
$$\text{Pilonidal Disease} = \text{Hair (H)} \times \text{Force (F)} \times \text{Vulnerability (V)}^2$$

The nature and variability of these causative elements have implications for persistent or recurrent disease. For example, the type and number of bacterial colonies present may be related to delayed wound healing following treatment [20]. Furthermore, deep tissue hypoxia is implicated in persistent pilonidal disease, with healing of complex wounds demonstrated by moving the suture lines to the open air [14]. These in turn have influenced various surgical treatment strategies

History and Physical

Patients presenting with pilonidal sinus often experience significant intergluteal cleft pain and occasionally drainage. Systemic signs, such as fever, are rare in immunocompetent patients. The pain experienced is often exquisite, preventing a patient from sitting or tolerating a physical exam.

A thorough history and physical examination are necessary to diagnose and create a treatment plan appropriately.



Evaluation

On clinical examination we have found there was two openings one in intergluteal cleft and second is on left side of gluteal region and there was a pus filled cavity about 5 cm away from primary sinus after MRI SINOGRAM report it was confirmed that it was case of pilonidal sinus

Treatment / Management

After taking consent from patient under local anaesthesia probing has been done with the help of copper probe and the whole track was identified. Then the opening was widened and all the hairs were removed from the track and then a proper drainage was ensured.

Ksharsutra was placed in the tract, antiseptic dressing and packing done with jatyadi taila. Patient was advised for regular hot sitz bath and cleaning of tracks with jatyadi taila two times a day.



Follow up and outcomes

Weekly follow up advised for Ksharsutra changing. The pus discharge was fluent in first week , track and cavity was gradually reduced and completely disappeared after 1months. Pain was also moderate in first week and later on gradually relieved. The discharge from the opening was also reduced gradually in 20 days and totally dried up in one month, and completed healing was achieved in 2 months after cut through. There was no complication seen during and after treatments and patient got free from all the symptoms. After 5 months of follow up, no recurrence is noted, patient was cured completed.



Conclusion

ksharsutra is a safe, effective and advanced technique which minimizes the postoperative time along with betterment in mild post procedural pain and minimum scar mark.

References

1. Hodges R. Pilonidal sinus. *Boston Med. Surg. J.* 1880;103 485–48. [[Google Scholar](#)]
2. Chinn B. Outpatient management of pilonidal disease. *Semin. Colon Rectal Surg.* 2003;14(4):pp166–172. [[Google Scholar](#)]
3. Buie L. Jeep disease. *South. Med. J.* 1944;37:103. [[Google Scholar](#)]
4. Ballas K., Psarras K., Rafailidis S. Interdigital pilonidal sinus in a hairdresser. *J. Hand Surg.* 2006;31(3):290–291. [[PubMed](#)] [[Google Scholar](#)]
5. Søndena K., Andersen E., Nesvik I.S.J. Patient characteristic and symptoms in chronic pilonidal sinus disease. *Int. J. Colorectal Dis.* 1995;10:39–42. [[PubMed](#)] [[Google Scholar](#)]
6. Ommer A., Iesalniaks I. The management of pilonidal sinus. *Dtsch Arztebl Int.* 2019 Jan 7;116(1–2):12–21. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
7. Bascom J. Pilonidal disease: origin from follicles of hairs and results of follicles removal as treatment. *Surgery.* 1980:567–572. [[PubMed](#)] [[Google Scholar](#)]
8. Notaro J.R. Management of recurrent pilonidal disease. *Semin. Colon Rectal Surg.* 2003;14(4):173–185. [[Google Scholar](#)]

9. Akinci O.F., Bozer M., Uzunkoy A. Incidence and aetiological factors in pilonidal sinus among Turkish soldiers. *Eur. J. Surg.* 1999;165(4):339–342. [[PubMed](#)] [[Google Scholar](#)]
10. Burney R. Treatment of pilonidal disease by minimal surgical excision under local anesthesia with healing by secondary intention: results in over 500 patients. *Surgery.* 2018;164(6):1217–1222. [[PubMed](#)] [[Google Scholar](#)]
11. Humphries A., Duncan J. Evaluation and management of pilonidal disease. *Surg. Clin.* 2010;90(1):113–124. [[PubMed](#)] [[Google Scholar](#)]
12. Karydakis G. Easy and successful treatment of pilonidal sinus after explanation of its causative process. *Aust. N. Z. J. Surg.* 1992;62(5):385–389. [[PubMed](#)] [[Google Scholar](#)]
13. Al-Khamis A., McCallum I., King P. Healing by primary versus secondary intention after surgical treatment for pilonidal sinus. *Cochrane Database Syst. Rev.* 2010;2010(1):CD06213. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
14. Karydakis G. New approach to the problem of pilonidal sinus. *Lancet.* 1973;302(7843):1414–1415. [[PubMed](#)] [[Google Scholar](#)]
15. Hull T.L., Wu J. Pilonidal disease. *Surg. Clin.* 2002;82:1169–1185. [[PubMed](#)] [[Google Scholar](#)]
16. Marks J., Harding K., Hughes L. Pilonidal sinus excision: healing by open granulation. *Br. J. Surg.* 1985;72(8):637–640. [[PubMed](#)] [[Google Scholar](#)]
17. Søndena K., Nesvik I., Andersen E. Bacteriology and complications of chronic pilonidal sinus treated with excision and primary suture. *Int. J. Colorectal Dis.* 1995 Jul;10(3):161–166. [[PubMed](#)] [[Google Scholar](#)]
18. Søndena K., Diab R., Nesvik I. Influence of failure of primary wound healing on subsequent recurrence of pilonidal sinus. Combined prospective study and randomised controlled trial. *Eur. J. Surg.* 2002 Nov 1;168(11):614–618. [[PubMed](#)] [[Google Scholar](#)]
19. Nelson J., Billingham R. *The ASCRS Textbook of Colon and Rectal Surgery.* Springer; New York, NY: 2007. Pilonidal disease and hidradenitis suppurativa; pp. 228–239. [[Google Scholar](#)]
20. Mark J., Harding K.G.H.L. Staphylococcal infection in open granulating wounds. *Br. J. Surg.* 1987;74:95–97. [[PubMed](#)] [[Google Scholar](#)]